Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION

1790 BROADWAY AT 58TH ST., NEW YORK, N. Y.

DR. J. HAMILTON CRAWFORD, Brooklyn, *Editor*

DR. CHARLES A. POINDEXTER, New York, *Associate Editor*

Vol. XII **March, 1943** **No. 3**

THE RELATION OF HEART SIZE TO PROGNOSIS

Cardiac enlargement and hypertrophy are reserve mechanisms whereby the heart is adapted to protracted strain so that it may maintain an adequate circulation. The mechanism of hypertrophy is less clearly understood than the physiological adaptations of the heart to transient stress. It is generally regarded that hypertrophy is preceded by some dilatation of the cardiac chambers. Increase in muscle fiber length, according to the Law of the Heart, results in increased force of contraction. While the physiologically dilated or hypertrophied heart may function with good mechanical efficiency, the true measure of cardiac, or more properly, circulatory efficiency, is the energy expenditure of the heart in maintaining its circulatory output, and not the ratio of the oxygen consumption to the kilogram-meters of actual work performed. Work expended against increased arterial resistance or to compensate for valvular defects is wasted in so far as the function of the heart to maintain cardiac output is concerned. Since oxygen consumption is proportional to fiber length, or diastolic volume, the cardiac output is maintained by the dilated or hypertrophied heart with greater energy expenditure, hence efficiency is decreased. Cardiac enlargement, therefore, betokens impaired cardiac reserve. Hypertrophy and dilatation as a rule are closely associated so that when the heart is enlarged, some degree of dilatation of the cardiac chambers is usually present with myocardial hypertrophy.

Roentgenologic and electrocardiographic examination both provide valuable information in the recognition of enlargement of the various cardiac chambers. Enlargement of the cardiac shadow as seen in the roentgenogram does not distinguish hypertrophy from dilatation. Since hypertrophy may be a matter of an increase in thickness of the myocardium of only a few millimeters, this cannot be discerned in the roentgenogram unless associated enlargement of the cavity is present. Concentric hypertrophy of the left ventricle, as occurs in hypertension, may be suggested, however, by increased convexity of the left ventricular curve even though the dimensions are not measurably increased. On the other hand, the characteristic electrocardiographic patterns which are observed in enlargement of the various cardiac chambers are the result of hypertrophy rather than enlargement as such.

Roentgenologic methods for the recognition of enlargement include fluoroscopy, orthodiagraphy and teleoroentgenography. Fluoroscopic examination of the individual cardiac chambers should always be carried out as a preliminary to further study. Fluoroscopy is not well suited for absolute measurement of the heart size because of distortion and enlargement of the cardiac shadow on the screen. Orthodiagraphy eliminates the factor of magnification by employing projection of the central ray on the fluoroscopic screen. Considerable training and exact application are required since there is a large personal equation. Roentgenograms made at six foot target-film distance eliminate most of the distortion due to divergence of the rays. At this distance there is approximately five per cent magnification of the cardiac shadow. Teleoroentgenography is the most widely employed method for estimating heart size. Roentgenograms should be made with the subject properly centered, at the end of a normal inspiration.

When it is not readily apparent whether the size of the cardiac shadow exceeds normal limits, measurements are a valuable diagnostic aid. Early studies by Moritz, Dietlen and others established the transverse diameter of the heart and the area of the frontal heart silhouette as the most useful of the cardiac measurements. The value of these measurements compared with others, such as the widely popularized cardiothoracic ratio, has been confirmed in more recent studies by Eyster[1], and by Kurtz[2]. The cardiac area is the most sensitive index of heart size, but the transverse diameter, as pointed out by Comeau and White[3], has the advantage of simplicity and greater precision in its measurement. Standards based on weight and height have been established for the transverse diameter both for the orthodiagram (Hodges and Eyster[4]) and the teleoroentgenogram (Ungerleider and Clark[5]). Measurement of the transverse diameter of the heart and the frontal cardiac area in relation to standards based on weight and height suffice for the estimation of heart size.

The cardiac area may be calculated accurately from the product, $\frac{\pi}{4}$ long times broad diameters which may be employed conveniently in lieu of planimetry. A nomogram chart has been prepared by Ungerleider and Gubner[6] which permits the frontal area to be read directly from the long and broad diameters. Another useful measurement is the transverse diameter of the vascular pedicle (frontal aortic arch silhouette), related to standards of weight and height just as the transverse diameter of the heart, as proposed by Sheridan[6]. A small correction for age is necessary for the aortic arch transverse diameter but not for the heart diameters. Increase in the aortic arch transverse diameter does not distinguish tortuosity from dilatation of the aorta. Values exceeding ten percent above that predicted from weight and height for the frontal area, transverse diameter of the heart, and transverse diameter of the frontal aortic arch may be considered abnormal, as few normal subjects fall outside this range.

Most types of heart disease impose a chronic strain on one or more cardiac chambers and cardiac enlargement is a usual accompaniment of the various types of heart disease. Since the degree of enlargement depends on the duration and the severity of the underlying lesion, there is in general a close correlation between prognosis and the heart size. There are some exceptions to this association, so that it is necessary to consider cardiac enlargement in terms of the individual types of heart disease.

Hypertensive and Aortic Valvular Disease. The left ventricle is the chamber most frequently affected

by heart disease. Left ventricular enlargement occurs in hypertensive heart disease, luetic aortic insufficiency, and in rheumatic heart disease where the aortic valve is involved either by insufficiency or stenosis. Left ventricular enlargement of variable degree may be found in mitral insufficiency, but the left ventricle is not enlarged when mitral stenosis predominates and, in fact, may be rather small. Congenital cardiac lesions, such as subisthmic aortic stenosis and coarctation of the aorta, also cause left ventricular enlargement. The degree of enlargement is directly related to prognosis in all these lesions which cause left ventricular strain, since the enlargement reflects the severity and duration of the lesion. However, it is not at all rare to observe tremendous left ventricular enlargement, particularly in cases with aortic insufficiency, where the subjects manifest no evidence of impaired cardiac reserve clinically and may be entirely unaware that they have heart disease. While such subjects may carry on normal activity for years, the course is rapidly downhill when cardiac decompensation ensues. It has been suggested that digitalis be administered prophylactically in subjects with chronic left ventricular strain in order to improve cardiac efficiency and forestall progressive enlargement in some measure. Even lesser degrees of left ventricular hypertrophy which may be evident only in the electrocardiogram while the roentgenogram is quite normal, carry an unfavorable prognosis. Thus in a recent mortality study in hypertension carried out by Daley, Ungerleider, and Gubner[7] it was found that there was a distinct increase in mortality with progression in the electrocardiogram toward an abnormal pattern. The life expectancy among those with the pattern of left ventricular hypertrophy was about half that of subjects with identical blood pressure readings in whom the electrocardiograms were normal. In a recent study by Gubner and Ungerleider[8] on Electrocardiographic Criteria of Left Ventricular Hypertrophy and Factors Determining the Evolution of the Electrocardiographic Pattern, the criteria proposed were left axis deviation in association with any of the following:

a) Increased amplitude of the QRS complex, best expressed as a sum of the R wave in lead 1 and S wave in lead 3 exceeding 2.5 millivolts.
b) Any perceptible depression of the ST segment in lead 1, even of as slight degree as one-half millimeter (0.05 millivolt).
c) Flattening of the T wave in lead 1 to below 1.0 millimeter (0.1 millivolt), or further degrees of T wave abnormality in lead 1 (and 2).

In subjects of asthenic build the characteristic ST and T wave changes may occur in the absence of left axis deviation. The electrocardiogram was found to provide the most sensitive method for detecting left ventricular hypertrophy. Enlargement of the heart in hypertension is related to its duration rather than its degree. The heart may be but slightly enlarged in subjects with malignant nephrosclerosis despite extreme hypertension because of the rapid course of the disease.

Rheumatic Mitral Valvular Disease. Prognosis in rheumatic heart disease depends perhaps more on reactivation of the rheumatic process than on the chronic strain imposed by the valvular lesions, but it is accepted to a greater degree than formerly that serious consequences may attend the mechanical handicaps imposed by valvular lesions independent of rheumatic activity. The chamber upon which greatest strain is placed in mitral stenosis and insufficiency is the left atrium. Enlargement of this chamber is not apparent in the postero-anterior view unless marked, and the degree of left atrial enlargement is best studied in the right anterior oblique position with the esophagus outlined by Barium. Enlargement of the right ventricle is usually found in advanced mitral disease. Pulmonary congestion and symptoms of severe congestive failure may be present in mitral stenosis when the heart is not markedly enlarged. This is due to obstruction of blood flow into the left ventricle rather than to true heart failure but the clinical picture and consequences are the same. Conversely, remarkable enlargement of the heart may be compatible with relative freedom from symptoms of impaired cardiac reserve, particularly in massive enlargement of the left atrium, where the contrast between the enormous heart size and comparatively mild clinical picture constitutes a definite syndrome. The other cardiac chambers, too, are markedly enlarged. This lesion develops most frequently in subjects in the third and fourth decade with a longstanding history of rheumatic heart disease. Auricular fibrillation is invariably present. Free mitral insufficiency, rather than mitral stenosis, is the underlying lesion, and characteristically a loud, long systolic murmur is heard over the precordium with no diastolic element. The enlarged left atrium displaces the esophagus back to the spine, elevates and compresses the main bronchi, extends to the right and may come to occupy the entire right lower thoracic cavity. Occasionally a marked systolic heave may be palpated over the right lower anterior chest due to systolic distension of the enlarged atrium resulting from free mitral regurgitation. The systolic distension of the left atrium can be observed in roentgenkymograms which also show large amplitude of the left ventricular contractions with very rapid filling in early diastole from the distended left atrium. The freedom from pulmonary congestion and frequently mild clinical picture these patients present may possibly be due to the enormously dilated left atrium acting as a reservoir between the heart and lungs to prevent pulmonary congestion. Tricuspid valvular disease, likewise, acts to prevent pulmonary congestion, so that subjects with this lesion, too, may be relatively free from dyspnea and orthopnea despite tremendous cardiac enlargement. It must not be regarded, however, that the prognosis is good either in massive left atrium or tricuspid disease, since these lesions signify longstanding and advanced rheumatic heart disease, but it is of interest that such patients may carry on for some time in comparative comfort.

Arteriosclerotic Heart Disease. Prognosis in arteriosclerotic heart disease depends on the state of the coronary arteries and is only secondarily related to the heart size. The heart is not appreciably enlarged as a rule unless there has been antecedent hypertension, or in the presence of frank congestive failure, and in the relatively rare cases where a ventricular aneurysm develops following coronary occlusion. The heart size may be normal in the presence of definite pulmonary congestion, and even during the acute stage of infarction the heart may not be enlarged although there may be clinical evidence of severe congestive failure.

Harry E. Ungerleider, M.D.
Richard Gubner, M.D.
New York, N. Y.

BIBLIOGRAPHY

1. Eyster, J. A. E., Arch. Int. Med. 41:667, 1928.
2. Kurtz, C. M., Orthodiascopy. Macmillan, N. Y. 1937.
3. Comeau, W. J., and White, P. D., Am. J. Roentgenol. 47:665, 1942.
4. Hodges, P. C., and Eyster, J. A. E., Arch. Int. Med. 37:707, 1926.
5. Ungerleider, H. E., and Clark, C. P., Am. Heart J. 17:92, 1939.
6. Ungerleider, H. E., and Gubner, R., Am. Heart J. 24:494, 1942.
7. Daley, R. M., Ungerleider, H. E., and Gubner, R., Ass'n. Life Insurance Med. Dir. 28:18, 1941.
8. Gubner, R., and Ungerleider, H. E., Am. Heart Ass'n., June 5, 1942. Archives of Internal Med. in Press.
9. Sheridan, J. T., Ass'n. Life Insurance Med. Dir. 28:49, 1941.